

HAEMATEMESIS; ITS AETIOLOGY, PROGNOSIS AND
TREATMENT WITH AN ANALYSIS OF 188 CASES.

by

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Vomiting of blood is one of the commoner medical emergencies and is consequently a condition with which all physicians have fairly frequently to deal. Much has been written about it and there is still a good deal of disagreement, especially regarding its treatment.

It is proposed in this paper to discuss its aetiology, prognosis and treatment and to analyse a collection of cases. The cases to be considered are all those admitted because of haematemesis, to the Leicester Royal Infirmary between the beginning of 1932 and the end of February 1937. Only those patients will be included who were admitted because of recent haematemesis and not those who had melaena only, or those who gave a history of having vomited blood at some time, but who were admitted for other reasons. Cases complaining of melaena have been excluded because it is difficult to judge the severity of their bleeding.

The cases to be considered number 188.

Aetiology

From a study of the literature it would at first sight appear that there is considerable difference of opinion regarding the relative frequency of the various causes of haematemesis.

Gordon Taylor (16) has stressed this point and in confirmation quotes figures given by Pauchet of Paris and by Gutman and Demole (17). According to Pauchet 90% of cases of gross haematemesis concern gastro-duodenal ulceration and only 10.% can be attributed to hepatic, splenic or appendicular disease. Gutman and Demole maintain that gastric and duodenal ulcers are the cause of only 19%.

In spite of such figures as these, however, it is my view that most of the apparent discrepancies among the statistics on this subject depend mainly on the evidence which individual workers have considered necessary for a diagnosis of peptic ulcer.

The causes of haematemesis have been well set out by Charles Miller (34), and what he says may be summarised:

1. Injury

- (a) Violent vomiting
- (b) Blow on epigastrium.
- (c) Passage of stomach tube
- (d) Corrosive poisons

2. Multiple emboli leading to tissue damage and auto-digestion, due to

- (a) Mitral stenosis
- (b) Ulcerative endocarditis

3. Blockage of veins
4. Miliary T.B.
5. Cirrhosis of the liver
6. Raised blood pressure
7. Certain blood diseases
8. Syphilis producing gastric ulcer or portal obstruction
9. Chronic ulcer
10. Carcinoma of the stomach.

The above causes he says account for about half the cases, and the rest are due to acute ulcers which may be called erosions even if they are visible, and may be associated with gall bladder infection, oral sepsis, appendicitis or visceroptosis.

To this list must be added splenic anaemia, vomiting of swallowed blood, carcinoma of the oesophagus, leakage of an aneurysm into the oesophagus, and simple tumours of the stomach.

Cases of haematemesis following injury to the liver have been described (44, 20).

The majority of these causes are extremely rare and will not be considered further.

Bulmer (5 and 6) has collected a large series of cases admitted to the General Hospital, Birmingham, between the years 1902 and 1926, and again from 1926-31, and he classifies these as follows,

<u>Cause</u>	<u>1902-26</u>	<u>1926-31</u>	<u>Total</u>
Acute ulcers	218	39	257
Chronic ulcers	249	72	321
Cirrhosis of the liver	25	5	30
Cancer of stomach	7	3	10
Portal thrombosis	1	-	1
Melaena neonatorum	2	-	2
Cirrhosis carcinomatoma	-	1	1
Doubtful cases	<u>24</u>	<u>3</u>	<u>27</u>
Total:	<u>526</u>	<u>123</u>	<u>649</u>

In the 1902-26 series Bulmer gives no figures to indicate the ratio of gastric to duodenal ulcers, but the 1926-31 chronic ulcers are subdivided into:

Chronic G.U.	(X-ray, operation or autopsy confirmation)	Cases ... 24
Chronic G.U.	(clinical diagnosis without confirmation)	... 34
Chronic D.U.	(X-ray, operation or autopsy confirmation)	... 9
Chronic D.U.	(clinical diagnosis without confirmation)	... 5

Apart from these 33 proved ulcers, he does not give any explanation as to the basis on which the diagnosis of ulcer rested, but presumably the clinical history counted for a good deal.

Hellier (19) in an analysis of 303 cases admitted to the Leeds General Infirmary between 1921 and 1932 found that the cause was:

Proved ulcer	in	96 cases
Probable ulcer	in	106 "
Splenic anaemia	in	14 "
Cirrhosis	in	10 "
Carcinoma	in	5 "
Doubtful	in	72 "

The "probable ulcers" were those where the history was "strongly suggestive" but the X-ray examination was either negative or not done. Of the 202 proved and probable ulcers, 32 were duodenal, 119 were gastric, 17 were jejunal and 14 were "unclassified peptic ulcer".

It will be seen that here again the diagnosis often depended on the clinical history. Leaving out the cases of splenic anaemia, cirrhosis, and carcinoma, the presence of an ulcer was only proved in 96 out of 274 cases, i.e. 35%.

M.E. Shaw (42) who found no definite cause in 39% of 28 cases, points out that if chronic ulcer were the common cause of haematemesis, one would expect a high proportion to show radiological signs even after treatment for 3 weeks or so. Shaw expresses the view that

acute ulceration of the stomach or duodenum "is a common if not the commonest cause of haematemesis". This acute ulceration may be part of a gastritis or duodenitis and be associated with infection of appendix, gall bladder, tonsils, teeth or sinuses. He quotes Gutman and Demole who attributed 41% of their cases of haematemesis to secondary dyspepsias and gastritis, and found that gastric and duodenal ulcers were the cause in only 19%. An acute ulcer may be defined as an ulcer which does not usually penetrate the muscular layer of stomach or duodenum, and exhibits none of the pathological features of chronicity. Shaw suggests that it does not seem to matter very much whether we refer to the condition as gastritis or as acute ulceration. There may be erosion of the gastro-duodenal mucous membrane in gastritis and duodenitis, and if an erosion is big enough we may refer to it as an acute ulcer.

Stewart (25) regards the terms "erosion" and "acute ulcer" as more or less synonymous. He suggests that if the term "erosion" is to be retained at all, it ought to be used merely as a synonym for a very superficial acute ulcer, and "haemorrhagic erosion" for such an ulcer in its earliest stages before complete digestion of the blood infiltrated slough. Acute ulcers are usually multiple (24).

The importance of gastritis has been much stressed by Continental workers, particularly by Faber (14) who believes that chronic gastro-duodenitis can produce typical ulcer symptoms without being complicated by the presence of any ulcer. Also he says (15) that fresh haematemesis or melaena appears to be a very frequent symptom in gastritis, and he quotes Torben Anderson (45), Nicolaysen (35) and Dahl (11) in support of this view.

It is interesting to note that, as long ago as 1901 when gastric diagnosis was not helped by X-rays, Hale White (18) concluded from autopsy evidence that many cases of haematemesis were due to a diffuse capillary oozing - gastrostasis as it was called. X/

And even longer ago than that, in 1815, Latour of Orleans in his *Histoire Philosophique et Medical des Hemorragies* (30) noted the possibility of fatal haemorrhage from the stomach without any obvious lesion.

C.G. Shaw (41) reported the causes in a series of cases of haematemesis as follows:

- 32 Gastric ulcer
- 16 Duodenal ulcer
- 38 other causes including gastritis.

And Gordon Taylor (1) found that among 124 cases of haematemesis admitted to the Middlesex Hospital in the years 1924-33 there were 20 deaths and 47 cases in which an ulcer was found by X-rays, so that an ulcer was proved in 67 cases out of 124.

In both these series therefore a definite ulcer was found in about half the cases.

On the other hand according to Rivers and Wilbur (39) it is almost always possible to prove a definite cause for haematemesis. In a series of 668 cases admitted to the Mayo Clinic they found only 2.2% in which the cause was not determined. By far the commonest cause was duodenal ulcer, 53.3%, only 6.4% being due to gastric ulcer.

Apparently, though they do not make this quite clear, most of the cases classified as ulcer were confirmed either by X-ray or operation.

It must be remembered that the "acute" cases of haematemesis such as are admitted to the ordinary hospital probably represent only a small proportion of this series.

Abrahams (1) concludes that the most common cause is an acute or chronic peptic ulcer, and this includes cases in which there is no previous history or where there has been irregular dyspepsia due to chronic

appendicitis - an acute ulcer also being present.

In the series of 188 cases which I have collected, the causes were as follows:

Carcinoma of the stomach	4 cases	2.1%
Splenic anaemia	4 "	2.1%
Cirrhosis of the liver	12 "	6.4%
Peptic ulcer or no cause found	<u>168</u> "	<u>89.4%</u>
	188	100.0%

The last group of 168 cases can be subdivided into:

Gastric ulcer proved by subsequent or very recent X-ray, or autopsy	...	20
Duodenal ulcer similarly proved	...	17
? Gastric ulcer: that is where the X-ray examination showed a doubtful ulcer...		19
? Duodenal ulcer: similar X-ray finding		13
? Stomal ulcer: similar X-ray finding		3
X-ray examination showed no lesion ...		54
No X-ray examination while in hospital and no autopsy	...	<u>42</u>
		168

This does not include acute ulcers found post mortem.

These will be referred to later.

Some of the cases classed as having no lesion, had delay in emptying of the stomach. Thus in 126 cases in this group, in which X-ray or autopsy examin-

ation was carried out, a definite ulcer was found in 37 cases, a doubtful ulcer in 35, and no *chronic ulcer* in 54. In other words even if we count the doubtful ulcers with the definite ones, we only find a lesion in 72 cases out of 126, i.e. a little over half.

The time during which symptoms of indigestion had been present before the haematemesis for which the patient was admitted, was investigated, and in the 168 cases classified under the heading "Ulcer or no cause found", the following was the result:

<u>Duration of Symptoms</u>	<u>No. of cases</u>
No previous symptoms	15
Less than 7 days	13
1 week to 6 weeks	22
6 weeks to 1 year	28
More than 1 year	87
No history given in Case Notes	3

It will be seen that about half the cases had a history of less than a year, and the other half of over a year. The indigestion in most of these latter had lasted for several years.

It would appear from a study of these figures that haematemesis is most likely to occur either within 6 weeks of the onset of indigestion, or after it has lasted for several years.

Most of the patients with cirrhosis of the liver had had some indigestion for months or years.

It will be noticed that the clinical history has not been taken into account in classifying the cause of haematemesis. It was felt that the history is not a reliable diagnostic point in this connection, and this was borne out by an examination of the case sheets. There seemed to be little connection between the type of symptoms or their duration and the X-ray findings. Many patients who had had indigestion for years showed no ulcer when X-rayed 3 or 4 weeks after a haematemesis, while others who had suffered only for a short time had an ulcer. Chiesman (8) also makes this point, but Hurst and Ryle have (48) suggested that the frequent X-ray findings following haematemesis may be due to acceleration in the healing of an ulcer after haemorrhage.

In this connection it was thought worth while to note the length of time which elapsed between the bleeding and the X-ray, and this was as follows:

Less than 2 weeks in	3 cases
2 - 3 weeks	25 "
3 - 4 weeks	41 "
Over 4 weeks (usually under 6 weeks	<u>40</u> "

It was not found that a positive result was found more often when the patient was X-rayed early as will be seen from the table below.

X-rayed before 25th day:	Positive	-	33	cases
" " " "	Negative	-	22	"
" after " "	Positive	-	30	"
" " " "	Negative	-	24	"

Positive results in this table include definite and doubtful ulcers, and these were evenly divided.

There does not seem to be any reason why a patient should not be X-rayed 3 weeks after a haematemesis provided he has regained enough strength to stand.

Test Meal Results

Excluding the cases of carcinoma, cirrhosis, and splenic anaemia, test meals were carried out in 90 patients with the following results:

Normal acidity	in	21
Hyperchlorhydria	in	45
Hypochlorhydria	in	19
Achlorhydria	in	5

Hyperchlorhydria is consistent with chronic ulcer or gastritis, but the large number of cases, 24 (26.6%), showing reduced acidity, was thought worth investigating further.

Of these 24 cases it was found that all had been X-rayed and that:

- 2 had had a ~~gastroenterostomy~~ gastroenterostomy.
- 2 had a gastric ulcer
- 2 had a doubtful gastric ulcer
- 18 showed no ulcer by X-ray.

The length of history of these cases with reduced acidity was then gone into and it was found that this was:

Under 1 month	in	7 cases
1 to 2 months	in	5 "
1 to 2 years	in	2 "
Many years	in	8 "

Plus 2 cases who had had gastroenterostomies.

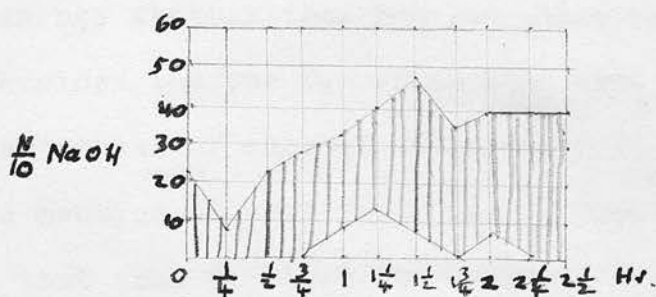
In other words the history was short in a much larger proportion of cases than it was in the whole series of cases. The explanation is probably that the patients with a short history had had an acute gastritis which had led to a hypochlorhydria or achlorhydria, (Faber) from which they had not recovered at the time of the test meal.

The patients with many years history and reduced acidity may have had a chronic atrophic gastritis.

So far as it goes the test meal evidence seems to be consistent with the view that gastritis is an important factor, and suggests that chronic atrophic gastritis must also be remembered as a cause.

Acidity was considered to be normal when the free H.C l curve lay within the area shown on the chart below. When the curve lay above or below it hyperchlorhydria or hypochlorhydria respectively was diagnosed.

Small variations from the "normal" were disregarded.



Burger and Hartfall (7) only found reduced acidity in 6 out of 52 test meals after haematemesis.

Previous Haematemeses

It is well recognised that some unfortunate patients suffer from haematemesis on more than one occasion. Thus Bulmer (6) in his 1926-31 series of 111 cases found 14 who had vomited blood previously.

Among the Leicester cases 4 of the 12 patients with cirrhosis of the liver had had a previous bleeding; while in the 168 cases belonging to the "ulcer or no cause found" group no less than 34 had had one or more previous haematemeses and 4 were found who have had a subsequent one.

Patients who had had an attack only a week or so before admission were not included. Many of the previous

attacks were small ones for which admission to hospital was not necessary, but it is probable that the figure 34 is too small, for this reason. As will be described later many of the cases of this series have been followed up, and those who were seen were asked among other things whether they had had other vomitings of blood besides the one for which they were admitted. The case sheets of many of those who said they had, bore no mention of this fact, and it therefore seems likely that some of those who have not been seen in the follow up had had previous haematemeses which have not been recorded.

It appears therefore that, leaving out the cirrhosis of the liver cases, at least 38 out of 168 or about 22.5% have had more than one attack of vomiting blood.

Babey (47) reported 39% of cases of haemorrhage from ulcer with a previous haemorrhage, but often this occurred only a few days before.

Considering the enormous number of people who suffer from gastric disorders and the comparatively small proportion of them who have haematemesis it seems to me rather significant that such a high percentage of them as this should have it more than once. It is said that almost all chronic peptic ulcers bleed to some extent sooner or later, but it has been shown, as I

believe, that chronic ulcer is the cause of haematemesis in only a proportion of cases - perhaps less than half. Why should some patients with gastritis or acute ulcer vomit blood on several occasions while many others with the same condition never vomit blood at all? Possibly it is mere chance and depends on the proximity of the inflammatory process to a large blood vessel; but it may be that the people who are prone to vomit blood have some other peculiarity which is responsible. I think the latter explanation is the more likely.

Abrahams (1) in a discussion at the Royal Society of Medicine in 1934 said "Undoubtedly certain individuals with ulcer tend to have a special tendency to bleed.

And Hurst (26) has said the same thing, and suggests that it may sometimes be a familial peculiarity.

Haematemesis in patients who have had a gastroenterostomy.

Hurst (23,29) has pointed out the frequency of haematemesis after gastro-enterostomy, and insists that the operation should not be done with a view to preventing a possible haematemesis in the future. He states that of 26 cases of haematemesis or melaena admitted to New Lodge Clinic, 11 or 42% occurred after operation (29). Hurst quotes Balfour (3) who found 13% of cases operated on at the Mayo Clinic who had

bled before, bled after operation: only 0.9% had bleeding after the operation who had not bled before. Hohlbaum (22) reported in 1922 that 20% of patients who had a gastroenterostomy for duodenal ulcer bled sooner or later afterwards.

It must be remembered that these figures refer to a time when gastroenterostomy was performed with perhaps too much enthusiasm.

Bulmer (5) found only 12 cases in 526 who had had the operation, but Wilkie (46) has stated that among cases operated on by him for a bleeding ulcer a third had a return of the bleeding subsequently.

In the series I have collected, 12 patients had previously had a gastroenterostomy. Unfortunately a formidable proportion of people continue to suffer from indigestion after gastroenterostomy, and I think that it is clear that if a patient continues to have indigestion after a gastroenterostomy he is quite likely to have a haematemesis, especially if he had one before his operation. It is said that if a jejunal ulcer forms it is particularly apt to bleed, and it may well be true to say that a patient with chronic indigestion is more apt to have a haematemesis if he has had a gastroenterostomy than if he has not.

Bleeding may also occur at any time after a

gastrectomy, but it is only of recent years that this operation has become popular, and it is too early to say how often it is followed at some time by haematemesis.

Cirrhosis of the Liver

Haematemesis is a common symptom of cirrhosis of the liver and is said usually to be due to dilatation of the veins at the lower end of the oesophagus. This disease accounted for 12 of the 188 Leicester cases, i.e. 6.4%, and only cases were included in whom the diagnosis was pretty certain.

Bulmer (5 and 6) taking his two series together found cirrhosis in 4.6%. Gutman (17) in 5%, and Rivers and Wilbur (39) in 2%, but definite cases of cirrhosis would probably not be sent to the Mayo Clinic.

Hellier (19) found this disease in 3.3%.

Although it has been suggested (Shaw, 42) that cirrhosis is a commoner cause of haematemesis than has been thought, there is no evidence for this, and we may conclude that cirrhosis of the liver is an important but not a common cause of haematemesis, accounting for something like 5% of the cases.

Haematemesis associated with Splenomegaly

The association of some cases of haematemesis with enlargement of the spleen has been recognised for many years, and was first pointed out by Morgagni.

It is not proposed to enter into a discussion regarding the pathology of this syndrome or the cause of the bleeding. It will be sufficient to say that there were 4 of these cases in the series, i.e. 2.1%. Hellier's (19) figure was 4.6% (14 out of 30); Bulmer does not mention the condition at all; Smith & Faber (43) have described a group of children showing splenomegaly, early haematemesis, and a blood picture which was normal except after the haematemesis, when there was a low haemoglobin anaemia. They point out that after the bleeding the spleen is reduced in size and may not be palpable, but as the blood is regenerated, the spleen increases in size and becomes palpable. The cases were followed up and it was found that they did not progress to cirrhosis of the liver and ascites.

The writers suggest that the condition is due to thrombophlebitis of the portal or splenic vein.

Carcinoma of the Stomach

As has been frequently pointed out carcinoma of the stomach seldom gives rise to a frank haematemesis, and it is therefore not surprising that this

disease accounted for only 4, i.e. 2.1% of the cases in the series; and that in none of these was a very large amount of blood lost.

Bulmer's (5 and 6) figure was 10 out of 649 and that is 1.5%. Gutman 3%, Hellier 5 out of 303 cases, or 1.6%.

Balfour (3) found gross haematemesis in 8% of the cases of carcinoma of the stomach coming to operation at the Mayo Clinic.

We may conclude that 1 to 3% of cases of haematemesis are due to carcinoma of the stomach.

Sex and Age

Of the 188 Leicester cases 113 were male and 75 were female, giving a ratio of M : F :: 1.5 : 1.

Hellier (19) found in 274 cases not due to cirrhosis, carcinoma or splenic anaemia that there were 189 males, and 85 females.

In 137 "ulcer" cases collected by Burger and Hartfall there were 95 males and 42 females.

Bulmer (5) in his 1902-26 series of ulcer cases found 160 males and 307 females, but in his 1926-31 ulcer cases (6), there were 80 males and 31 females.

It is difficult to explain the variation here, but it is probably true to say that haematemesis is considerably more common in men than it is in women.

There is no doubt that haematemesis due to cirrhosis of the liver is most common in men. Of the 12 cases I have collected 10 were male, and 2 female.

The average age of the whole series of cases was worked out and was found to be 44.3 years. That of the fatal cases was 52.4.

The ages in the Leicester cases varied from 9 to 80, but a large number of patients were between 30 and 55.

These findings agree closely with those of Aitken (2) who in his 262 cases found the average age to be as follows:

Patients who recovered with medical treatment ...	41
" " died " " " ...	51
Patients who were treated medically and by blood transfusion ...	41
Patients treated surgically ...	45

Burger and Hartfall (7) also found that the average age was about 40.

Proportion of Gastric to Duodenal ulcers

It is generally held that where a haematemesis is due to a chronic ulcer it is much more likely to be due to a gastric than a duodenal one.

This belief is not borne out by the Leicester cases, in which there were 20 proved cases of chronic gastric ulcer and 17 of proved chronic duodenal ulcer.

Other published results show a marked gastric ulcer preponderance as will be seen from the table.

<u>Reference</u>	<u>Gastric</u>	<u>Duodenal</u>
Bulmer (6) Proved ulcers	24	9
Hellier (19) Proved and probable ulcers	119	32
Shaw (41)	32	16
Rivers & Wilbur (39)	53.1%	6.4%
Burger & Hartfall (7) confirmed ulcers	41	15
Babey (47)	66%	26%

It is difficult to explain this discrepancy, but it has been suggested by others that gastric ulcer is not a much commoner cause than duodenal, and the position must be regarded as doubtful.

Discussion

There are a large number of very rare causes of haematemesis: these have been already mentioned.

There are three causes, cirrhosis of the liver, splenic anaemia, and carcinoma of the stomach, which are not common. Together they probably account for about 10% of cases. The rest are due to chronic or

acute ulceration or erosion of the stomach, or duodenum or jejunum.

Radiology has advanced so much in recent years that when we get a negative report from a competent radiologist, I think we must assume that it is almost certain that no ulcer is present.

Taking this into account and remembering the various results described above, it seems likely that chronic peptic ulcer is the cause in not more than half the cases, - perhaps less. The rest must be due to acute ulceration or erosions.

In my opinion it is misleading to say that acute ulcer is the cause in these remaining cases. In the first place, while the actual loss of mucous membrane which has led to the bleeding may be of recent origin, a generalised inflammation may have been present for many years, and in the second place "acute ulcers" are usually multiple. (Stewart, 24).

There is no real distinction between an acute ulcer and an erosion - except possibly as regards depth; and it seems to me more reasonable to suppose that the majority of these remaining cases are due to a gastritis or gastroduodenitis which we can label acute or chronic according to the length of history.

Very likely an acute exacerbation is necessary in the chronic cases to bring about a haematemesis.

It would seem therefore that gastritis (or gastro duodenitis) and chronic peptic ulcer are the common causes of haematemesis, and that the former is as common or nearly as common as the latter.

Prognosis

Mortality

Many series of cases have been published to indicate the mortality rate in haematemesis, and these have been compiled from various sources. Some of them are based on post mortem room records, others on clinical records, and of the latter some give prognosis in cases of haematemesis, while others give the prognosis in cases of haematemesis due to ulcer.

These differing sources of information no doubt account to some extent, as Paterson (37) and Burger and Hartfall (7) have pointed out, for the variability of the conclusions.

Hurst has for long maintained that haemorrhage from gastric or duodenal ulcer is seldom fatal, and he bases his opinion on the following points: (27 & 48)

- (a) In only 2 out of 350 private cases of ulcer had death resulted from haemorrhage (1929).

- (b) From post mortem statistics at Guy's Hospital between 1911 and 1920, recorded by Conybeare, the mortality rate of cases admitted for haemorrhage appeared to be 2.5%.
- (c) At Leeds it was found by Stewart that death by haemorrhage from a gastric or duodenal ulcer was found in 0.41% of 9,000 consecutive autopsies.
- (d) Hurst quotes Crohm of New York who he says published similar statistics.
- (e) The number of death from haemorrhage at Guys between 1919 and 1935 was 4 out of 82 cases of chronic gastric, duodenal or anastomatic ulcers i.e. 4.8%.
- (f) Ryle found a mortality rate of 3.1% in private cases of severe haematemesis.

Hurst, after inquiry from a number of General Practitioners, concluded that not more than 2.6% died. We are concerned here, **however**, with cases severe enough to be admitted to hospital.

Now as regards point (a) one can make no criticism. Point (b) seems the less convincing because it is based on post portem and not clinical case records, and **our** faith must be severely shaken when we read the paper of Burger and Hartfall (7).

They report 177 cases of haematemesis admitted to Guys Hospital in the next ten years, i.e. 1921-30. 137 cases were due to ulcer and the mortality among them was 22.6%, or excluding cases treated surgically 18%.

Point (c) does not give us any indication of the prognosis in haematemesis.

As regards point (d) Crohn (9) reports 101 cases of severe haemorrhage in patients with ulcers. 7 of these were operated on with 5 deaths, and 94 were treated medically with 4 deaths. Presumably the severe cases were operated on and the true mortality rate is 9 in 101, or 9%, and not 4 in 94 or 4.2% as Hurst suggests.

Crohn quotes others including Nielson (36) who consider that the mortality rate is low.

Point (f) is important but we must note that it refers only to cases of chronic ulcer with haemorrhage, and so it presumably includes melaena.

One other worker of importance who has reported a low mortality rate must be mentioned, and that is Meulengracht. Treating his patients with unrestricted diet he has reported (33) a series of 286 cases. Leaving out 19 cases of cancer (with 3 deaths), and 16 of cirrhosis with 3 deaths, he was left with 251 cases of whom 3 died, giving a mortality rate of 1.5%. Leaving out one patient who died before treatment was

started the rate was 1%, but 1.5% seems the truer figure.

Other statistics are much less encouraging.

Bulmer (5 and 6) found 8.3% deaths from acute ulcers and 11.6% from chronic ulcers in the 1902-26 series, and he found 13.5% mortality from haemorrhage in all types of ulcers in the 1926-31 series.

Aitken (2) reported 27 deaths in 255 cases of haematemesis admitted to the London Hospital between 1929 and 1933: a rate of 11%. He tried to distinguish the grave cases among whom 3 out of 7 died. Grave cases were those in whom the R.B.C. count was less than two million and the haemoglobin less than 40%, and those who clinically were gravely ill.

Hellier (19) found a death rate of 14.8% in cases of proved or probable ulcer: and in 274 cases of haematemesis not due to splenic anaemia, carcinoma or cirrhosis the rate was 13.1%.

C.G. Shaw (41) had 5 deaths in 35 cases of haematemesis treated conservatively, i.e. 14.3%, and Davies and Nevin (12) found death from haemorrhage occurred in 21.5% of 391 cases of significant haematemesis or gross melaema. They also try to distinguish severe cases in whom the rate was 45.1%.

Cullinan & Price's (10) figure was 18.1% in 105 cases of haematemesis. Hinton's (21) figure was

20%. Gordon Taylor (1) reported a rate of 24% in cases of haematemesis from supposed ulcer admitted to the Middlesex Hospital.

And finally Chiesman (8) found that 25% of cases with gross haematemesis or melaena died.

Among the 188 Leicester cases of haematemesis there were 30 deaths which may be classified as follows:

Chronic gastric ulcer confirmed P.M.	...	5
Chronic gastric ulcer confirmed by X-ray previously	...	1
Chronic duodenal ulcer confirmed P.M.	...	2
Chronic duodenal ulcer confirmed by X-ray previously	...	1
Acute ulceration confirmed P.M.	...	8
Cirrhosis of the liver confirmed P.M.	...	5
Cirrhosis of liver not confirmed P.M.	...	2
Splenic anaemia confirmed P.M.	...	1
No P.M. and no definite diagnosis	...	<u>5</u>

30

1 case had a chronic perforated gastric ulcer.

1 case with a chronic gastric ulcer had perforation of the transverse colon, and one case with an acute gastric ulceration had a perforated gangrenous appendix.

In each of these three cases there was general peritonitis which was presumably the cause of death.

If we leave out these three cases the death rate from haematemesis is 14.6%

If we leave out the three perforations and all the cases of cirrhosis, splenic anaemia, and carcinoma we are left with 19 deaths in 165 cases, i.e. 11.5%.

It may be noted at this point that judging from the case records it is evident that the large majority of the cases had had severe haematemeses.

Several other points from the post mortem records must be noted.

First, in 4 of the 7 chronic ulcers an open blood vessel was seen.

Secondly, of the 8 cases with acute ulceration, there were 2 or more ulcers in 6.

Thirdly, of the 9 chronic ulcers confirmed by autopsy or recent X-ray, 6 were gastric and 3 duodenal.

Finally, in many of the records it was stated whether or not blood was found in the stomach and intestines, and it was found that in only one case (of cirrhosis) was there blood in the stomach, and in a great many cases it was not present in the small intestine.

This point will be referred to later when treatment is considered.

The time which elapsed between admission and death varied from a few hours to 28 days, the average being 7 days. Five patients died within 24 hours of admission, and most of these had started treatment at home before they were sent to hospital.

The average age of the patients who died was 52 and there were 19 males and 11 females.

The mortality rate of the men was therefore 19 out of 113, i.e. 16.8., and that of the women 11 out of 75, or 14.6%. The male and female death rates were thus very much the same. Others, e.g. Burger and Hartfall (7) and Bulmer (5 and 6) have found the male rate about three times the female.

It will be noticed that the cirrhosis of the liver cases did very badly. There were 12 cases with 6 deaths.

Discussion

It will be seen that the mortality rate in the Leicester cases is better than some and worse than others of the various series quoted. If we disregard for the moment the possibilities of Meulengracht's treatment, and consider cases of haematemesis of a severity requiring admission to hospital, I think we may conclude that,

- (1) The mortality rate in haematemesis from all causes is a good deal higher than it is in haematemesis from the common causes - peptic ulcer and gastritis.
- (2) The mortality rate from all causes is about 15.4% or perhaps more.
- (3) The mortality rate in haematemesis from peptic ulcer and gastritis is at least 10%.

Ultimate Prognosis

In order to find out how patients with haematemesis get on after they leave hospital a "follow up" investigation was carried out. Patients admitted in 1936 and 1937 were not investigated because it was thought that too short a time had elapsed for their subsequent history to be of any value. All surviving patients however who were admitted from 1932-35 inclusive were written to, excluding those diagnosed as carcinoma, cirrhosis or splenic anaemia. These numbered 110, and information was obtained in 76 of them, or 69%. Almost all of these 76 were actually seen, but a few were unable to come and sent their story by letter. The questions asked them were:

- (1) How long had they had indigestion before admission: - this was done to check the case sheet records.

- (2) Had they had other attacks of haematemesis before or since.
- (3) How long did they take to get back their normal strength after the haematemesis.
- (4) How much indigestion, if any, had they had since.

The subjects of questions one and two have already been dealt with; and it may be said in passing that the answer to question 1 almost always agreed with the case notes.

As regards question 3 it was of course difficult for these people to remember the answer, and the results must therefore be taken with a good deal of reserve, but on the whole, it was surprising how often, after a little thought, the patient was able to give a fairly definite reply. The results of this investigation are given below.

<u>Period</u>	<u>No. of Cases</u>
1 month	3
2 "	8
3 "	10
4 "	8
5 "	1
6 "	13
7 "	1
8 "	3
10 "	1
1 year	9
18 months	4
Never got back to normal	1
Could not remember	<u>14</u>

Thus 21 cases were normal in less than 3 months, 22 in from 3 to 6 months, and 19 in over 6 months.

It is not known of course what treatment these patients received during these periods, and possibly some of them did not get as much iron as they should have. As far as these figures go, however, they suggest that about half the patients of this series were below par for 6 months or more after their attack of vomiting.

The amount of indigestion suffered by these patients after their discharge from hospital is indicated below:

No indigestion at all: No dietary precautions	...	9 cases
No indigestion at all if diet adhered to	...	14 "
Hardly any indigestion	...	15 "
Occasional indigestion	...	17 "
Frequent indigestion	...	16 "
Very bad indigestion	...	5 "

The term indigestion was used to indicate any of the usual gastric symptoms.

Most of the patients in the last four groups dieted to some extent, and most of the patients who had indigestion before or after their stay in hospital stated that they were now better than they were before their haematemesis.

Of the cases who had had a haematemesis after a short period of symptoms, some were symptomless, but many had some degree of indigestion. This point was rather striking and it appeared therefore, that quite a number of patients with indigestion of some years duration had a haematemesis a few weeks or months after it started.

It will be seen that only 9 out of 76 patients were perfectly normal, and that only 38 patients were almost normal, or symptomless if they dieted.

This investigation suggests that among individuals who have a haematemesis, very few of them are likely to regain perfect health, and about half of them will have a significant degree of symptoms of indigestion afterwards, in spite of dieting.

Treatment

Most writers on this subject agree that the essentials of medical treatment are rest, morphia and perhaps atropine, a preliminary period of starvation, rectal salines, and if necessary blood transfusion.

Morphia and Atropine.

Varying amounts of morphia are suggested. Thus Hurst (28) thinks gr.1/6 is enough, while Bolton (4) recommends gr.1/3 - $\frac{1}{2}$.

Bulmer (5) suggests that possibly we are inclined to give too much morphia.

Atropine is given by some on the grounds that it inhibits gastric secretion (28).

Practically all the Leicester cases were given one or more injections of morphia, and about half were given atropine. There is no doubt about the value of morphia given in sufficiently large doses to prevent restlessness. The value of larger doses of morphia, e.g. gr. $\frac{1}{2}$ and of atropine is more doubtful.

Most of the Leicester cases were given morphia gr. $\frac{1}{4}$, and it was often repeated several times.

Period of Starvation

There is some difference of opinion as to the length of this period. Thus Maclean (31) in 1928 advised nothing by mouth for 4 days; Hurst (29) nothing by mouth till bleeding has been stopped for 48 hours; Izod Bennett (1), nothing by mouth for 36-48 hours; Bolton (4), teaspoonfuls of water after 12 hours, and albumin water and whey when the bleeding has been stopped for 2-3 days; while Meulangracht (33) feeds his patients from the start.

There seems, even apart from Meulangracht, to have been a tendency to shorten the period of starvation.

It is I think well established that the mortality rate, in patients who have a second haematemesis soon after the first, is very high - 78% according to Gordon Taylor (1), and further that patients who have a haematemesis and then stop bleeding seldom die. The difficulty then, is to know how to treat the patients who go on bleeding; and if we decide to start feeding a certain time after bleeding has stopped, these people may never get anything by mouth at all.

Meulangracht (33) treats his cases with plenty of varied food from the first day, and gives the following reasons for doing so.

- (1) Exhausted patients often died after haemorrhage in spite of scrupulous dieting.
- (2) Sometimes patients with protracted haemorrhage stopped bleeding when they were given food.
- (3) Very often ambulant patients recovered from severe melaena without making any particular change in their ordinary diet.
- (4) It seemed a questionable advantage to starve a patient at a time when he is presumably in special need of support.
- (5) It was doubtful whether it was really desirable that the stomach should be empty of food and contain free acid gastric juice.

(6) It seemed improbable that a diet insufficient in calories and vitamins was ideal as a means of promoting healing of ulcers.

Meulangracht might also have mentioned that many patients being treated for haematemesis are given very little fluid, even when they are having rectal salines, for surely insufficient fluid intake cannot help a patient who has lost a large quantity of blood.

It was pointed out when prognosis was being considered that the period between haematemesis and death averaged about a week. It was also pointed out that post mortems seldom showed blood in or near the stomach, which suggests that bleeding usually ceases some time before death: (though blood can pass down the alimentary canal extremely rapidly). Taking all this into consideration I think we must conclude that lack of food and drink often plays a large part in producing a fatal result.

It should be noted however that Hurst and Ryle (48), in a paper published a few months ago, concluded that there was no evidence to show that malnutrition was a factor in any of the fatal cases of a series analysed.

It was not possible to discover the exact details of treatment in most of the cases of this series, but about 10 cases treated during the last six months of the period, were given water, or milk and water, from the first day, starting usually 5 or 6 hours after admission. The details of one of these cases is as follows:

Female, aged 75: seemed very ill on admission. Vomited blood 6 hours ago. Starting 2 hours after admission she was given,

1st day	:	$\frac{1}{2}$ oz. citrated milk $\frac{1}{2}$ oz. water	hourly
2nd "		1 oz. citrated milk 1 oz. water	"
3rd "		2 oz. citrated milk	"
4th "		3 oz. citrated milk	2 hourly
5th & 6th day		6 oz. citrated milk	"
7th - 9th "		8 oz. citrated milk	"
10th day		Solid food added	

In addition she was given a teaspoonful of water during the first few days whenever she complained of thirst, but she had no rectal salines. This patient recovered.

Of the 10 patients treated in this way, one died, and I think this type of treatment should be tried out more extensively.

Details of Meulangracht's method of treatment will not be given here. It will be sufficient to say that from the day after their admission to hospital they are given alkalis and iron, and a full puree diet of varied food, and they are allowed as much food as they want.

His mortality rate was 1 or 1.5%, and a figure of 1% was also found by Rischel (38) using similar treatment.

These results seem to be exceedingly good and it will be interesting to see how successful the treatment is in other hands.

Schiodt (40) has published records of 10 cases treated by what he calls the "ulcer cure", and 10 by Meulangracht's method. He showed that blood was regenerated more quickly in the second group, but it should be observed that they were given iron and the others were not.

Rectal Salines

It is frequently advised that rectal salines should be given, and a daily rectal wash out also.

Hurst (28) advises 15 oz. of 4% dextrose solution per rectum alternately with 15 oz. of saline every 6 hours. Bolton (4) gives 10 oz. of 5% glucose saline per rectum four-hourly and also subcutaneous

salines if thirst is acute.

Hurst and Ryle (48) say that half to three quarters of a pint of normal saline should be given 4-hourly, the foot of the bed being raised.

I think these writers are somewhat optimistic here: and I doubt if it is often possible to have such large amounts of fluid retained by the patient.

The main disadvantage of giving rectal salines is that it may be followed by further bleeding. This has happened on several occasions in my experience, and most of the cases treated at Leicester with fluids from the first day were given nothing by the bowel and sometimes did not have their bowels open for a week.

Alkalis from the start are sometimes recommended. Hurst (28) advised washing out the stomach with ice cold water and this may be theoretically sound but is not to be lightly undertaken. Burger and Hartfall (7) reported that they could find a record of only one case of haematemesis at Guys Hospital between 1921 and 1930 who had been treated in this way, and in his recent articles Hurst (47,48) does not mention the treatment.

Various "haemastatic" substances have been advised: iron perchloride, adrenaline, calcium, and haemostatic sera, but it is probable that these are of no value.

Blood Transfusion

Abrahams (1) at the end of a discussion on haematemesis in 1934 stated that there seemed to be general agreement that blood transfusion was helpful in certain cases. This is no doubt the case, but Christiansen (49) found the mortality to be doubled in the Kommune-hospitalet, Copenhagen after transfusion became common.

According to Bolton (4) there is no danger of transfusion leading to repeated haemorrhage, but Hurst & Ryle (48), have uttered a word of warning and urge that transfusion should not usually be given unless the haemoglobin percentage is below 30, and the patient appears to be in danger of death from anaemia. They think that 10 oz. is enough, and transfusion should not be repeated unless haemorrhage recurs.

Blood transfusion by the continuous drip method (32) described by Marriot and Kekwick may be of value, and must be further investigated.

Many of the Leicester cases received transfusion and two of these were given by the drip method. It is extremely difficult to assess the value of transfusion to these patients, but I think that it should probably be carried out in most patients whose haemo-

globin is below 30%, and in a few with a haemoglobin above 30%, the determining factor being the clinical condition.

Operation

Opinions regarding the desirability of urgent operation in cases of haematemesis from a chronic ulcer, vary from that of Finsterer (50), who regards immediate operation as imperative, to that of Hurst and Ryle (48) who think that operation should be reserved for "the very rare cases in which it seems probable that the source of bleeding is a large hole in a sclerotic vessel exposed at the base of a chronic ulcer".

Gordon Taylor (16) is a believer in operation, and gives certain indications for surgery -

1. Terrific bleeding
2. Haemorrhage with perforation.
3. Drip transfusion failing to control the haemoglobin.
4. Operate when the drip has raised the haemoglobin to 90% in a man and 80% in a woman, in cases:
 - (a) Where recent X-ray showed a deep ulcer
 - (b) With long and definite ulcer history
 - (c) With pyloric or duodenal stenosis.

(a),(b),(c) are indications whatever the extent of the bleeding may be.

- (d) With midgastric narrowing sometimes
- (e) With a second haemorrhage within a year with a probable ulcer
- (f) With severe bleeding where ulcer cannot be eliminated, sometimes
- (g) Where carcinoma is definitely suspected.

Gordon Taylor urges the importance of early operation, and the value of the drip transfusion. He agrees that operation is almost never indicated unless a chronic ulcer is present. The majority of writers seem to agree with Abrahams (1) who stated that the indications for surgery were rare.

If operation is performed, it should have as its object the stoppage of the bleeding, and nothing else should be attempted. None of the Leicester cases were treated surgically.

Even with further experience of operation combined with drip transfusion it will be difficult to decide the part which surgery should play in this condition, and at present it is impossible to do so.

One thing however, can be said: consideration of the evidence regarding prognosis in severe haematemesis does seem to indicate that the mortality is by no means negligible; and therefore any method of treatment which may tend to reduce the number of deaths, must be very carefully examined.

Summary

1. 188 cases of haematemesis have been analysed.
2. It is concluded that about 10% of cases are due to cirrhosis of the liver, splenic anaemia, carcinoma of the stomach, and rare causes.
3. The other 90% are due to
 - (a) Chronic peptic ulcer
 - (b) Gastritis (or gastroduodenitis)and these two causes are probably equally common. The gastritis may be acute or chronic.
4. It seems likely that many patients who have a haematemesis possess some peculiarity which makes them prone to bleed.
5. Haematemesis is very liable to take place after gastroenterostomy, especially if the patient bled before the operation.
6. Haematemesis is more common in men than in women.
7. The average age of patients suffering from haematemesis is about 40.
8. In cases of a severity requiring admission to hospital the immediate mortality in haematemesis from all causes is about 15%, or possibly more: in haematemesis from peptic ulcer and gastritis is not less than 10%.

9. In the series of cases analysed, half of the patients took 6 months or more to get back their normal strength, very few of them regained perfect health and about half continued to have a significant degree of indigestion in spite of diet.
10. It is suggested that lack of food and drink may be a factor sometimes in causing death. Fluids were given by mouth to a few of the cases in this series, starting a few hours after admission. This method of treatment might be tried out further.
11. If this treatment is adopted it is probably wiser not to give rectal salines.
12. Blood transfusion should usually be carried out if the haemoglobin is under 30% and occasionally when it is over 30%, the determining factor being the clinical condition.
13. Immediate operation is of no value except in cases of chronic ulcer, and in them its value is doubtful. Further evidence regarding treatment by operation combined with drip transfusion is desirable.

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